STATISTICAL ANALYSIS

During the past 16 years we have made direct microscopic examinations or cultures in 614 patients with inflammatory eruptions of the hands as presenting symptoms. Of these, 9.2 per cent revealed the presence of fungi of the trichophyton group, 2.5 per cent showed monilia, leaving 88.3 per cent which were negative. This group of cases included the following diagnoses: "Eczema," 186 cases; phytid eruptions, 63 cases (in all of these the feet were positive and the hands negative); infectious eczematoid dermatitis, 82 cases; dermatitis venenata, 195 cases; dermatophytosis, 56 cases; erosio interdigitalis, 13 cases; moniliasis of the palm, 1 case; and hyperkeratotic dermatitis, 16 cases.

SUMMARY

Diagnosis of fungus infection of the hand is being made indiscriminately and without adequate evidence.

A diagnosis of a fungus infection of the hand in the majority of instances automatically excludes a case from further consideration as an occupational compensable problem, often with a gross miscarriage of justice.

The differential diagnosis of inflammatory eruptions of the hands constitutes a very complex dermatological problem, which can only be solved by a careful history, experienced clinical examination and confirmatory laboratory procedures.

A statistical analysis of 614 private patients, with eruptions of the hands as presenting symptoms in whom microscopic examination of scales or vesicles was made, revealed that only 11.7 per cent were due to an actual infection with fungi or yeast.

Without confirmatory evidence or dermatological experience, a diagnosis of fungus infection of the hand stands an 88 per cent chance of being wrong. Even with a great deal of dermatological clinical experience, a positive diagnosis of fungus infection of the hand is very difficult without laboratory evidence.

2007 Wilshire Boulevard.

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The public believes, and I am afraid we have led them to believe that we have considerable power in the control of influenza and poliomyelitis, when as a matter of fact the procedures that we now employ in these two diseases are of no demonstrated value. In German measles and chickenpox far too much ineffective energy is being wasted for fear the public will interpret our lack of action as wilful neglect rather than lack of scientific knowledge. In the case of whooping cough more facts are needed before we can serve a very helpful purpose.—JOHN L. RICE, M.D., *Commissioner of Health, New York City.*

HYPERTENSION AND THE SURGICAL KIDNEY*

THOMAS E. GIBSON, M. D.
San Francisco

INTRODUCTION.—The close relation between renal disease and hypertension has been recognized since the time of Bright[1] more than a century ago, but only since 1934 has convincing experimental evidence regarding the possible mechanism of this relation appeared.

Prior to 1934 there had been innumerable clinical and anatomical studies which served gradually to distinguish between two entities: (1) The primary nephropathies with secondary elevation of blood pressure, and (2) "essential hypertension," in which high blood pressure preceded by long periods the development of any other clinical or functional evidence of renal impairment. With respect to essential hypertension, it was clearly demonstrated by clinicopathological correlation that practically all of these patients, at post-mortem, showed extensive arteriolar disease, particularly within the kidneys; but argument continued for decades over the precedence of the hypertension or the arteriolar degeneration (Mallory).

EXPERIMENTAL EVIDENCE

In 1934 attention was suddenly shifted in a more profitable direction by Goldblatt and his associates[2] in the publication of a series of experiments on the production of hypertension and its physiological mechanism. He found that, if a clamp is placed on the renal artery which constricts but does not occlude it, the blood pressure rises significantly for a period of weeks. If the clamp is removed, or the ischemic kidney removed, the blood pressure returns to normal. *The hypertension is, therefore, clearly dependent upon the presence of ischemic renal tissue.* Bilateral constriction of the renal arteries, or unilateral constriction of one renal artery and opposite nephrectomy, produces severe and permanent hypertension. These experiments were widely confirmed, and other methods of inducing renal ischemia appeared, such as exposure of the kidneys to x-rays (Hartman),[3] and enveloping them in a bag of cellophane, which provokes the development of a dense fibrous constricting capsule (Page).[4] Removal of the encapsulated kidney promptly restored normal blood pressure, just as in the Goldblatt experiments.

A wealth of experimental data is now accumulating in an effort to elucidate the *mechanism* of

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* Read at the California Medical Association Meeting, Del Monte, May 5-8, 1941.

† However, Peet[7] who recognizes renal ischemia as the basic factor in hypertension and has obtained cure or marked relief of hypertension in a large percentage of cases by splanchnicectomy and lower dorsal ganglionectomy, bases the rationale of his procedure on the relief of renal ischemia by interruption of the sympathetic vasoconstrictor outflow to the kidneys. It would seem in humans that nervous as well as endocrine factors may serve at least to maintain the vascular tree in a reactive state to the stimulus which provokes hypertension.

hypertension thus produced. The work of Page and his associate[5] and Collins[6] demonstrates clearly that the nervous system is not directly involved in the genesis of renal hypertension, since renal denervation, total sympathectomy, or total destruction of the spinal cord by pithing does not prevent its development.†

Elimination of a nervous mechanism immediately suggested the possibility of a *humoral* mechanism as responsible in the form of some pressor substance elaborated by the ischemic kidney. Much experimental and clinical evidence points to the fact that a substance extractable from the renal parenchyma (Helmer and Page)[8, 9] called "renin" is actually liberated by the ischemic kidney, and is the cause of the hypertension. However, renin in itself is not a pressor substance, but requires chemical activation to produce vasoconstriction. This "renin activator" reacts with renin to produce "angiotonin," which appears to be the actual vasoconstrictive or pressor substance.

The recent work of Kohlstaedt and Page[10] indicates that the essential cause of renin liberation in the ischemic kidney is reduced pulse pressure. According to this theory, compression of the renal artery "leads to partial conversion of pulsate to continuous bloodflow in the kidney, with edema and anoxia of the cells of the tubules as the chief results. Increase in cellular membrane permeability follows and allows the liberation of the large renin molecule. Renin reacts with renin activator to produce angiotonin, which itself raises blood pressure and causes efferent glomerular arteriolar constriction and further tubular anoxia. A vicious circle may be thus set up, which results in *sustained arterial* hypertension." Moreover, the renin angiotonin relation is a double one, since renin not only forms angiotonin, but with further contact will destroy it. The possible antigenic properties of homologous renin and angiotonin are now under investigation.

From the clinical standpoint, one of the most perplexing questions arising at the present time concerns the explanation of the fact that if a given lesion of the kidney causes hypertension, why are not such lesions associated with hypertension in all cases? Why, in some instances, is the hypertension of moderate degree, and extreme in others? And finally, why does nephrectomy definitely and permanently relieve the hypertension in some cases and not in others? The application of the experimental data to human lesions is far from clear, but certain conclusions may be drawn. Evidence is accumulating to show that the organism possesses potent and effective mechanisms for the prevention of vasoconstrictor substances. Studies on the antipressor or inhibitor mechanisms are being carried out at the present time. Angiotonin is apparently not an end product which in tself causes hypertension, but it, in turn, requires an activator to become effective. Presumably, activators may be exhausted or counterbalanced by the development of inhibitors, so that hypertension may not develop in some cases of a given renal lesion. Hypertension, when present, may not be relieved by removal of the diseased kidney even though caused by it, provided it has been present for too long a period, and arteriolar sclerosis has become generalized, involving the opposite kidney.

CLINICAL EVIDENCE

With this experimental background as a basis, let us now consider the clinical problem. It was not long before Goldblatt's experimental observations were confirmed by clinical reports of cases in which the blood pressure of patients with hypertension returned to normal after removal of a diseased kidney. These reports seemed to prove that unilateral renal lesions may cause hypertension, and that removal of the affected kidney is often followed by recovery. Review of the current literature indicates that an extensive variety of lesions of the urinary tract have been found to be associated with hypertension, and that they have been cured by nephrectomy or less radical surgical procedures in a significant proportion of cases. These lesions may be classified in three general groups:

I. Gross vascular lesions of the renal artery or its branches.
- a. Trauma with infarction.
- b. Thrombosis
- c. Polycystic disease
- d. Tumors, (adenocarcinoma, Wilms' tumor)
- e. Ectopic kidney
- f. Aneurism
- g. Atheromatous placques

II. The obstructive uropathies.
- a. Hydronephrosis
- b. Ureteral obstructions
- c. Bladder neck and urethal obstructions
- d. Renal calculus disease

III. Chronic Inflammatory Lesions.
- a. Chronic atrophic pyelonephritis
- b. Chronic bilateral pyelonephritis
- c. Sclerosing perinephritis
- d. Renal tuberculosis
- e. Periarteritis nodosa

The basic factor causing hypertension in these uropathies appears to be the occurrence of renal ischemia, just as in experimentally-induced renal hypertension. Limitation of space precludes a detailed analysis of all the clinical reports of hypertension cured or relieved by nephrectomy. Obviously, hypertension is not a concomitant of all surgical lesions of the kidney. One of the most pertinent factual studies of the incidence of hypertension in surgical renal lesions is that of Braasch, Walters, and Hammer.[11] They found that the incidence of hypertension, in a group of 1,684 patients subjected to renal surgical operation, was no higher than it was in a group of patients taken at random. In this group the surgical lesions most often associated with hypertension were atrophic pyelonephritis. Hypertension afflicted 46.5 per cent of these patients. The incidence of hypertension was low in cases of pyelonephritis without atrophy and sclerosis. Acute cortical renal infection, or perinephric abscess, was seldom a factor in causing hypertension.

Hypertension was observed in 20.3 per cent of cases operated for renal calculus. Hypertension in these cases was four times as common when the stone was associated with infection. However, the deciding factor was not the degree of infec-

tion, but the amount of vascular sclerosis and parenchymal atrophy.

Hypertension was noted in 14 per cent of cases of hydronephrosis. As with stone, the hypertension was related to the degree of tissue atrophy and vascular sclerosis, rather than to the size of the hydronephrosis.

Hypertension was present in 7.6 per cent of cases of renal tuberculosis.

Hypertension was found in 27.7 per cent of cases operated for adenocarcinoma.

They found further that hypertension may result after a conservative renal operation as a result of nephrosclerosis, and that the blood pressure returns to normal after removal of the affected kidney.

There appears to be no uniform relation between renal function and blood pressure. They found that, in most cases of hypertension, there was no evidence of reduced function and, conversely, patients whose renal function was reduced often had no hypertension. Furthermore bilateral renal involvement, such as frequently occurs in renal lithiasis, hydronephrosis and tuberculosis, was not an etiologic factor in hypertension. However, Braasch[12] states, in another article dealing with bilateral pyelonephritis, hypertension was found twice as often in cases of impaired renal function as in those of normal renal function, and the incidence of hypertension roughly parallels the duration and severity of the disease.

They conclude that hypertension will be relieved by nephrectomy in about 70 per cent of cases in which it accompanies atrophic pyelonephritis, in 50 per cent of cases in which it is associated with renal tuberculosis, and in about 25 per cent of cases in which it is an accompaniment of renal stone, hydronephrosis or tumor.

In a review of 198 patients with hypertension subjected to renal surgery, the blood pressure became normal in one third of the cases and remained normal for more than a year.

The conclusions of Crabtree and Chaset[13] are pertinent at this point. They made a careful histologic study of 150 cases representing severe unilateral renal damage which were subjected to nephrectomy. An attempt to correlate hypertension and renal vessel change met with failure. Three cases of hypertension showed no alteration in renal vessels. Elevation of blood pressure was not the rule, even in pyelonephritis where vascular changes were marked, and nephrectomy was not followed by appreciable reduction in blood pressure readings before operation. They conclude that the exact etiologic factor in renal (ischemic) hypertension is as yet unknown. The pathologic and anatomic elements seem less important than an as yet unknown physiologic element. Evidence is not produced by this study to encourage employment of nephrectomy in hypertensive cases, except for recognized surgical indications.

These conclusions seem at variance with those expressed in the monographic contribution on pyelonephritis of Weiss and Parker[14] who found a definite correlation between vascular changes and hypertension. However, they studied for the most part cases of severe bilateral pyelonephritis, and recognized that, in unilateral pyelonephritis with advanced vascular changes, hypertension may or may not be present. They estimate that 15 to 20 per cent of cases of malignant hypertension are caused by pyelonephritis.

One of the most striking examples of relief of hypertension by nephrectomy was related to us last year by Leon Howard[15] at the Western Branch Urological Meeting in Victoria. He reported the case of a five-year-old girl with malignant hypertension, her blood pressure going as high as 200/150. At operation, he found an aneurism of the left renal artery. Following nephrectomy, the child's blood pressure promptly returned to normal and has remained normal. This is but one of many striking cases which have been reported, involving a variety of surgical renal lesions both in children and in adults. The clinical demonstration that such casual relationships do exist has opened up a new field of investigation, and has shown the necessity of complete urological investigation of all patients with hypertension, even in the absence of a history of kidney disease, or urinary findings suggesting disease of the urinary tract. However, until knowledge of the mechanism of hypertension is more complete, the present enthusiasm for nephrectomy in hypertension must be tempered by a careful consideration of the criteria for nephrectomy which have guided us in the past. There must be a clear-cut indication for nephrectomy regardless of the associated hypertension, as illustrated in the following personal case:

REPORT OF CASE

CASE 1.—Male, age 32, entered the Southern Pacific Hospital in August, 1938. The medical staff feared malignant hypertension, since his blood pressure, even after bed-rest, stayed at 225 systolic and 145 diastolic. Urological study revealed a tuberculous left testicle and epididymis, and silent occluded left renal tuberculosis. These were removed. The kidney was about three times normal size and consisted of a thin-walled septate sac, filled solidly with caseous material weighing 450 grams. On the day following operation his blood pressure was 140/90 and two weeks later 135/80. Two years later he is well and active, blood pressure 145/95.

COMMENT

Renal function may show no impairment in the earlier phases of essential and malignant hypertension, as demonstrated by urea clearance and urine concentration tests, which constitute our most sensitive clinical tests. Yet, the presence of functional disturbance can be demonstrated by coincidental tests of diodrast and inulin clearance, as shown by Homer Smith.[16] Plasma clearance of diodrast offers a method of measuring the rate of bloodflow through the kidneys, whereas the rate of filtration of water from blood in the glomeruli can be measured by inulin clearance. These tests reveal the presence of constriction of

the efferent arterioles of the glomeruli long before concentrating power and urea clearance are affected. It has been demonstrated that the specific action of angiotonin, which is present in the blood of patients with essential hypertension, is the production of efferent arteriolar spasm. Thus the current trend of investigation indicates that all hypertension, even essential hypertension, may be due to renal ischemia. This phase of the problem has been discussed at length in a recent publication by Corcoran and Page.[17] They conclude that the endocrine system (particularly the adrenal cortex and hypophysis) plays a secondary rôle in the causation of hypertension.

The endocrine system apparently serves to maintain the vascular tree in a state receptive to hypertensive stimuli, but does not participate in the mechanism causing hypertension. The nervous system presumably plays a similar rôle.

The last decade has seen the advent of important advances in the treatment of hypertension. Hitherto, treatment was medical, consisting in the main of palliation and sedation, which served only to modify the outcome in a small proportion of cases. The last few years have seen the development of the surgical treatment of hypertension, the history of which has been well reviewed by Martin.[18] The brilliant results achieved in large series of cases have served to place the surgical treatment of hypertension on a firm foundation. Operations on the sympathetic nervous system, consisting of splanchnicectomy and ganglionectomy, and more recently urological operations consisting of nephrectomy and correction of obstructive uropathies, have achieved many brilliant results. In the light of present knowledge, the choice of operation, neurosurgical or urological, must depend upon careful evaluation of factors in the individual case. Since renal ischemia is recognized as the basic factor in hypertension (Peet), our first concern should be a thorough urological appraisal of every case. In unilateral nephropathies, nephrectomy may give complete and permanent relief. In the obstructive uropathies the elimination of the obstructive factor may be the answer to the problem. In bilateral renal involvement, where the pathological changes are moderate or not clinically demonstrable, as they may be in essential hypertension, the treatment of choice is neurosurgical. In severe bilateral nephropathy, surgery so far offers little hope, and treatment must of necessity be medical.

CONCLUSIONS

1. Hypertension may result from kidney disease, either bilateral or unilateral.

2. Experimental evidence has shown that ischemia is the essential factor causing hypertension in kidney disease.

3. Renal ischemia results in the liberation from the kidney of a vasoconstrictor or pressor substance called renin. Renin alone causes no hypertension, but reacts with a Kinase-like substance in the blood stream referred to as renin-activator. The result is angiotonin, a highly-active pressor substance, which in turn appears to require an activator other than renin-activator. The body also possseses potent mechanisms for the prevention of vasoconstrictor action. Studies on the inhibitor mechanisms are being carried out at the present time.

4. A rapidly-increasing number of clinical observations have shown that the blood pressure of patients with hypertension may return to normal after removal of a diseased kidney. Analysis of clinical reports indicates that a great variety of kidney lesions may cause hypertension. These lesions fall into three general groups: (1) gross vascular lesions of the renal artery or its branches, (2) the obstructive uropathies, and (3) chronic inflammatory lesions. In all three groups clinical evidence supports the experimental in indicating ischemia as the important factor initiating the hypertension.

5. Chronic infection appears to be the most important single etiological factor responsible for renal ischemia. It is estimated that 15 to 20 per cent of cases of malignant hypertension are due to chronic pyelonephritis, even though in some cases the infection has run its course and is healed. Emphasis, therefore, should be placed upon the elimination of urinary tract infections in their early stages.

6. All patients with hypertension should be submitted to complete urological investigation as a part of their routine examination, even in the absence of signs or symptoms of urinary tract disease.

7. Reasonable expectancy of improvement or cure of hypertension of renal origin can be hoped for by appropriate treatment of the pathology thus revealed.

8. A personal case is reported in which hypertension was cured by removal of a silent, occluded tuberculous kidney. The urinary findings were normal, and there were no signs or symptoms of kidney disease. This case is cited to illustrate the importance of urologic study of all hypertensive patients, even in the absence of signs or symptoms pointing to disease of the urinary tract.

450 Sutter Street

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TUBERCULIN PATCH TEST: ITS RELIABILITY*

A COMPARISON WITH THE MANTOUX TEST

PETER COHEN, M. D.
Santa Barbara

VOLLMER and Goldberger,[1] in 1937, developed a tuberculin test, employing a tuberculin ointment incorporated in adhesive tape, to be applied to the surface of the skin as a means of identifying those individuals infected with the tubercle bacillus. As a first step, or "screening process," to select those for whom an x-ray of the chest is indicated in mass efforts to find new cases of tuberculosis, their method, known as the "patch test," has been discussed in numerous reports, most of them commendatory. Its obvious advantages over the common method of intradermal injection (Mantoux Test) either of Old Tuberculin or of the Purified Protein Derivative, has induced many clinics and health departments to compare the dependability and usefulness of these two methods.[2]

* From the Santa Barbara County Health Department, Santa Barbara, California.

In the case-finding program in Santa Barbara County, we have used the Vollmer-Lederle Patch Test and the Mantoux Test, employing first the highly-diluted Purified Protein Derivative (P.P.D.) followed by the stronger solution for those who showed no reaction to the first injection, as recommended by the National Tuberculosis Association. In 1939 and 1940, we applied these two tests simultaneously to 1535 unselected individuals, largely school children, from the kindergarten through junior college, but including also a few teachers.

PROCEDURE

With each group to be tested we began, on a Monday, by applying the Mantoux test, first strength, to the left forearm, and the patch test to the anterior or inner surface of the left arm after cleansing the skin with acetone. Close contact of the patch ointment with the skin was assured by firm rotating pressure with the thumbs. On the following Wednesday the patch was removed, the Mantoux test area was examined, and, if the Mantoux had produced no reaction, the second strength was given at a site near by. On Friday, the fourth day, the tests were "read" and the results recorded. Early in the following week (7th or 8th day) we ordinarily inspected the patch tests again in order to discover late reactions which had not yet developed at the time of the fourth day "reading".

TABLE 1.—*Results: Reactions to Simultaneous Mantoux (P.P.D.) Tests and Patch Tests*

Class I.—Mantoux Negative	Number Tested	Patch Test Negative	Patch Test Positive
(a) Negative in *both* strengths	1283	1273	10
(b) First strength not given; second, negative	87	86	1
TOTAL:	1370	1359	11

Class II.—Mantoux Positive	Number Tested	Patch Test Negative	Patch Test Positive
(a) Negative in first strength, but positive in second	78	4	74
(b) Inconclusive in first strength, but positive in second	5	0	5
(c) First strength not used; second, positive	14	0	14
(d) Positive in first strength; second not administered	68	0	68
TOTAL:	165	4	161

COMMENT

Of those 1370 individuals who failed to give any reaction in the regular two-strength administrations of the Purified Protein Derivative Mantoux Test, we found 11 in whom the patch test gave positive results; while in only four instances (out of 1363) did we obtain a positive Mantoux when the patch was negative. In 1520 cases the results were the same for both tests, a correlation of 99 per cent.

It will be noted, further, that the patch test positive reaction did not fail in any individual in whom the Mantoux had indicated a high degree